Methamphetamine Associated Cardiomyopathy and Fatal Thrombus Formation

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Introduction:

Stimulant abuse is a rapidly growing epidemic both in the United States, and around the world. Reports show that nearly 4.7 million Americans have tried methamphetamines at least once. It is known that methamphetamines and related compounds significantly increase cardiovascular morbidity and mortality. Substance-related chronic inflammation and tissue fibrosis can cause irreversible cardiac structural changes, leading to conditions like methamphetamine-associated dilated cardiomyopathy (MACM). Left ventricular dilation results in hemodynamic flow disturbances, which promote turbulent blood flow and stagnation. Furthermore, the sympathomimetic effects of methamphetamines also disrupt pathways in coagulation homeostasis, providing further incitement of thrombus formation. This unstable catecholamine driven pathway is thought to create myocardial ischemia via direct free radical oxidation, mitochondrial injury, and metabolic derangements. This is thought to be the cause of left ventricular systolic dysfunction with apical ballooning and hypokinesis. Here, we report a case of large left ventricular thrombus formation in the setting of methamphetamine-associated cardiomyopathy.

Case Report:

Substance-related chronic inflammation and tissue fibrosis can cause irreversible cardiac structural changes, leading to conditions like methamphetamine associated dilated cardiomyopathy (MACM). Left ventricular dilation results in hemodynamic flow disturbances which promote turbulent blood flow and stagnation. Furthermore, the sympathomimetic effects of methamphetamines also disrupt pathways in coagulation homeostasis, providing further incitement of thrombus formation. Here, we report a case of large, left ventricular thrombus formation in the setting of methamphetamine associated cardiomyopathy and unfortunately resulted in flash pulmonary edema and acute systolic congestive heart failure with cardiogenic shock. A contrast CT scan of the chest revealed extensive bilateral pleural effusions and atelectasis. A 2.4 cm opacity was seen in the apex of the left ventricle suggesting the presence of a thrombus (Figure 1). Transthoracic echocardiogram identified an echogenic mass measuring 2.1×1.8×1.8 cm (Figure 2).

Conclusion:

Considering that amphetamine-containing products are listed by national reports as one of the most widely abused illicit substances, clinicians must be aware of the many complications associated with their use. Pertinent to the case presented, MACM is not an uncommon cardiac complication within this patient population. Methamphetamines play a significant causative role in numerous cardiac pathologies, including hypertension, tachyarrhythmias, coronary vasospasm, and cardiomyopathies. Abuse of centrally acting stimulants results in a state of sympathetic overdrive due to excessive catecholamine production. This response leads to uncontrolled hypertension, tachycardia, and stress induced cardiomyopathy (Takotsubo). Some cases have been reported of myocardial infarction without evidence of epicardial coronary stenosis, suggesting global coronary microvascular vasospasm and a diagnosis of myocardial infarction with non-obstructed coronary arteries (MINOCA). There are reports that suggest that the myocardial structural changes can be

reversible with cessation of methamphetamine use. Unfortunately, with persistent or prolonged use, these changes become irreversible, leading to ventricular dilation and fibrosis.
We have reviewed some of the potential cardiac complications of methamphetamine use, focusing on catecholamine-driven methamphetamine-associated dilated cardiomyopathy with associated thrombus formation. Currently, there are no guideline driven recommendations for methamphetamine user screening with 2D-TTE or for prophylactic anticoagulation for patients with dilated cardiomyopathy. Although this is the case, we would like to urge clinicians to become familiar with these potential complications and screen select patients with symptoms or who are at higher risk for complications. We would also like to emphasize the importance of obtaining a detailed social history and providing patient education on potential complications.

Figure 1: Contrast CT scan of the chest
A 2.4 cm opacity can be seen within the apex of the left ventricle (LV) consistent with thrombus formation. The LV thrombus can be seen in the sagittal (A), coronal (B) and axial (C) views.

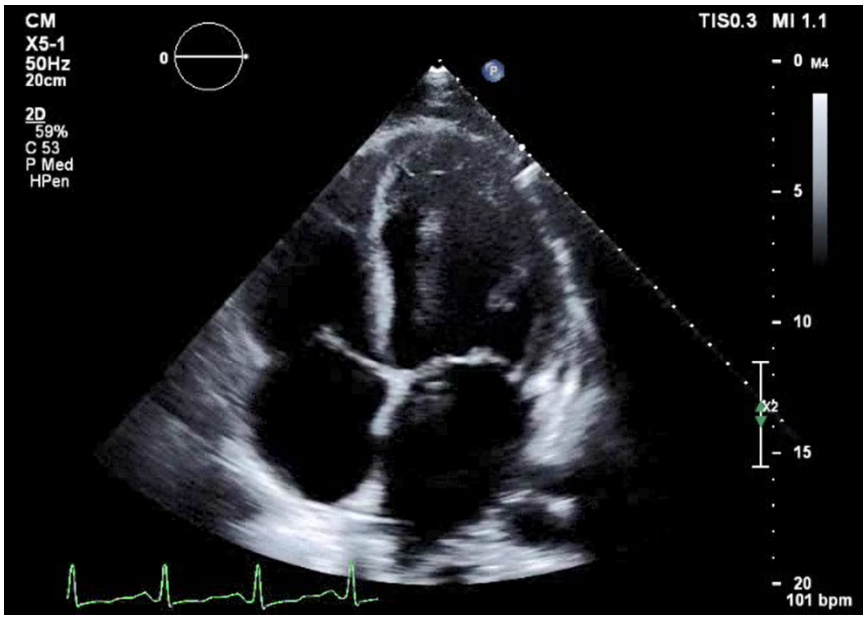

Figure 2: 2d-Transthoracic Echocardiography: A left ventricular apical thrombus can be seen on 2d-TTE in the 4-chamber apical view.